stained by fibrin stains, these semilunar deposits are highly characteristic of postmortem kidney sections and form the "microangiopathy" of MAHA. Studies of the correlation between the degree of microangiopathy and the severity of schistocytosis opened the way to the present understanding of the pathophysiologic features of this syndrome.

In animal experiments the entire process can be simulated by injection of a procoagulant enzyme such as arvin after first blocking the fibrinolytic system with epsilon amino caproic acid. The fragmentation of red cells by fibrin strands has also been studied and photographed *in vitro*.

The clinical picture is that of a hemolytic process superimposed upon the pathophysiology of the DIC and complicated yet further by changes resulting from the primary disease process. As a result treatment of MAHA is properly directed first at the underlying disease and secondarily at the DIC.

Those diseases most frequently involved are obstetrical disorders (abruptio placenta, retained dead fetus), the hemolytic uremic syndrome, malignant hypertension, polyarteritis nodosa, Gram-negative sepsis, and disseminated adenocarcinoma.

MAHA has considerable clinical relevance for it is easily recognized by examining a properly prepared blood film. When the typical schistocytes are present along with thrombocytopenia, DIC can be confidently diagnosed.

BRIAN S. BULL, M.D.

REFERENCES

Brain MC, Dacie JV, Hourihane DO'B: Microangiopathic haemolytic anaemia: The possible role of vascular lesions in pathogenesis. Brit J Haemat 8:358-379, Oct 1962

Bull BS, Brain MC: Experimental models of microangiopathic haemolytic anaemia. Proc Roy Soc Med 61:1134-1137, Nov 1968

McKay DG: Progress in disseminated intravascular coagulation. Calif Med 111:186-199, Sep 279-289, Oct 1969

Platelet Transfusions

Platelet transfusion therapy has progressed on the basis of new data in human immunogenetics and new technical advances yielding greatly increased availability of platelet concentrates (PC). The essential indication for platelet transfusions is still thrombocytopenic hemorrhage, and a careful hematological evaluation continues to be necessary to rule out complications, such as coagulopathy or disseminated intravascular coagulation, for which platelet transfusions could be ineffective or even contraindicated. The risks of transmission of hepatitis or cytomegalovirus are the same as for other blood components, and once platelet transfusion support is decided upon, platelet concentrates are the preparation of choice.

Each unit of PC produces, at about four hours post-transfusion in an adult, an increment in the recipient's platelet count of about 12,000 per cu mm. An adult with no autologous platelet production would thus require approximately four units of PC twice weekly to maintain a platelet count greater than 20,000 per cu mm. If hemorrhage occurs with platelet counts above that level, then another defect in hemostasis must be sought. Fever, sepsis, hepatosplenomegaly, and antiplatelet allo-immunity or auto-antibodies will all decrease platelet transfusion effectiveness; splenectomy will increase effectiveness; and androgens and prednisone (up to 100 mg a day) will not alter effectiveness. Currently, there are no clinically practical crossmatching tests for platelets. Rh differences have no effect, and ABO differences have a variable effect on recovery of transfused platelets. Whenever possible, rules of compatibility (for example, O→A, but not A→O) should be followed, but in an emergency, incompatibilities may be breached with little risk to the recipient (Note: This holds for PC only). For any multi-transfused patient population the clinically most significant antigens are those of the HL-A system. Multi-transfused patients become refractory to transfused platelets because of allo-immunity to foreign HL-A antigens at a median of about eight weeks. HL-A compatible platelets, however, can be tolerated indefinitely with no evidence of allo-immunization and no loss of transfusion efficacy. Utilizing known immunogenetic data for the HL-A system, each sibling of a patient will have a one-in-four chance of being HL-A compatible, compared with a one-in-fifteen-hundred chance for unrelated donors. In fact, bi-weekly four-unit plateletpheresis of a sibling donor has kept aplastic anemia or leukemia patients free of bleeding for periods as long as three years, and in several instances these compatible platelets had to be flown hundreds of

miles from donor to recipient each time. Patients who have a significant post-transfusion increment clearly benefit from platelet transfusions. There are, however, conflicting opinions and little evidence concerning the value of platelet transfusions in patients with ITP or allo-immunization sufficient to block any post-transfusion rise in platelet count. Nevertheless, in the presence of significant bleeding in either of those two problem cases, most hematologists would probably attempt a trial of platelet transfusions. Although compatible platelets could stop the bleeding in the allo-immune patient, to date there have been no platelet donors known to be compatible with ITP antiplatelet factors. Finally, the rules of immunogenetics have also been applied to iso-immune neonatal thrombocytopenic purpura, with plateletpheresis of the mother providing an excellent source of compatible platelets to protect the infant until his own platelets have recovered from the insult of the transplacentally acquired maternal antiplatelet antibodies.

F. C. GRUMET, M.D.

REFERENCES

Grumet FC: HL-A typing for platelet transfusion, *In* Proceedings 23rd Annual Meeting of the American Association of Blood Banks, San Francisco 1970, pp 65-72

Grumet FC, Yankee RA: Long-term platelet support of patients with aplastic anemia. Ann Intern Med 73:1-7, Jul 1970

Platelet Storage

Recent advances in techniques of processing platelets for transfusion have extended the blood bank storage life of this valuable blood product from approximately six hours to almost 72 hours. The significance of this prolongation of shelf life is that blood banks are now capable of providing platelet units at any time, day or night, without having to call in new donors. Blood collected during regular donor hours is drawn into a closed system of several interconnecting bags made of new plastics with ACD or CPD anticoagulant. Because platelets are sensitive to low temperatures, the freshly drawn unit of whole blood must be immediately centrifuged at room temperature (22°C). The packed red cells, after separation from the platelet-rich plasma (PRP), can then be kept at the standard 4°C blood storage temperature. (Logistically, the two-temperature requirement means that blood drawn in bloodmobiles and kept refrigerated in transit back to the processing center is far less suitable for extraction of platelets than is blood drawn at the center itself.) The PRP can be further centrifuged, again at 22°C, to provide a platelet concentrate (PC) with approximately 75 percent of the platelets of the original 500 ml of blood now in a volume of 15 to 50 ml. With no further additives or manipulations, the PC is stored at room temperature, with gentle agitation. The PC is then immediately available when needed for transfusion. The advantage of ready availability greatly outweighs the small loss of effectiveness incurred during the first three days of room temperature storage of PC. Techniques for more prolonged storage of platelets, either by freezing or by use of new additives, are currently under investigation in a number of laboratories.

F. C. GRUMET, M.D.

REFERENCE

Murphy S, Sayar SN, Gardner FH: Storage of platelet concentrates at 22° C. Blood 35:549-557, Apr 1970

Reed-Sternberg Cells in Non-Hodgkin's Disease

Reed-Sternberg cells (RSC) are not pathognomonic for Hodgkin's disease, but the diagnosis of Hodgkin's disease is not made in their absence. Recent reports confirm a previous, but seldom emphasized, observation that Reed-Sternberg cells mean Hodgkin's disease only when they are in association with the proper histopathologic background features, or milieu, of one of the sub-types of Hodgkin's disease.

Multinucleated cells resembling Reed-Sternberg cells have been described in a variety of reactive and neoplastic proliferations. A striking example is the presence of multinucleated cells, which may be indistinguishable from the diagnostic cells of Hodgkin's disease, in lymphoid tissue from persons with infectious mononucleosis. The cellular proliferation in tissue in infectious mononucleosis is predominantly that of an extraordinary number of plasma cell precursors